SOME OBSERVATIONS ON PLAGUE,

WITH AN ACCOUNT OF

SOME ORIGINAL WORK AND EXPERIMENTS,

by

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In the following pages some points connected with plague and the methods of combating it will be dealt with, such as have been suggested by an experience of many years.

Between the years 1899 and 1907 I worked through eleven epidemics of plague in India. I had full charge of the Civil Plague hospitals in Karachi and in Poona; as Civil Surgeon at Sholapur, Satara, Nasik, and Bj Bijapur, I had control of the arrangements for dealing with various outbreaks. Again, from 1904 to 1907 I was in charge of the laboratory connected with the Gold Fields of Mysore, and moreover it was my duty to deal with the epidemics which occurred during that period. From first to last over 1500 cases of plague came directly under my personal observation. All the experiments which I may have occasion to describe or allude to hereafter were performed by me in the above-named laboratory.

From the above it will be understood that my opportunities for studying the disease under many and varied conditions have been numerous. The material to hand for bacteriological as well as

therapeutical investigations was always abundant.

HISTORY.

The term Pestis has such a wide and varied significance that it is impossible to assert that the records which have come down to us from past centuries are those of the disease Plague as we know it today. Any disease which became a scourge was called "Plague": witness the "Plagues of Egypt"mentioned in Exodus. When Measles broke out in the Figi Islands in 1785,it carried off about fifty thousand of the inhabitants. Had this occurred centuries ago,there is no doubt but that it would have been called a "Pestis" and as such have been accepted today.

Early accounts,though of interest,do not throw much light on the disease which has ravaged India for the past fourteen years,and which seems more than likely to make itself painfully felt in other portions of the civilized world. Apart from biblical notices,one of the earliest records seems to be that made by Petavius,who states that the world was swept by a Pestis in 763 B.C.

Thucydides gives an account of the Athenian plague in 430 B.C.,but it is questionable if this disease was bubonic plague.

Pliny tells of an outbreak in the Isles of Greece in 187 B.C.

Galen speaks of one in Rome in the second century A.D.

Strabo,quoting Posidonius,tells of an outbreak in Spain; but what is of greater interest is that he states that there was great mortality among mice,and that"the inhabitants soon connected the death of rodents with the scourge which afflicted them from time to time,and they moved to other parts. "

Eusebius writes about the great plague in Syria in 302 A.D.

The Justinian Plague is mentioned by more than one author. It swept over Italy,and it would seem that it never really died out but continued right on into the next century. There is a note of the death of one Vighard,Archbishop elect,who had gone to Rome to have his appointment confirmed by Pope Vitalianus in 670. Vighard and all his suite died from the disease.

In England some form of plague swept over the country in the year 430 A.D. It is not until 664 A.D.,however,that we find records of a disease in England which in any way compares with that which swept over the continent in the previous century. In its nature,and the degree of devastation which it caused,it resembles it very closely.

Weymouth,Bristol,London and Oxford all became infected in 1348,and Stow mentions numerous other outbreaks which carry us on to the great Plague of London in 1664-5. After that,England seems to have escaped.

On the Continent Plague was more or less endemic right up to the commencement of the nineteenth century, and it is chiefly the countries bathed by the Mediterranean that suffered. Marseilles in 1720 lost 60,000 of her inhabitants. Syria was decimated in ~~16~~ 1760, Egypt in 1792; and in the neighbouring countries the disease was endemic throughout the century.
It is supposed to have died out about the year 1850, but I have it on the authority of some medical men who have lived all their lives in Asia Minor that Plague has practically always been endemic there.

These early records do not teach us much, but they emphasize the fact that it was always the sea-port towns or those situated on river banks which became infected first and suffered the most.
In Asia there are several centres which are marked down as endemic, and it is from these that the present outbreaks have spread ~~all~~ over the world.

Having thus passed in rapid review some of the most important dates in the history of Plague epidemics, I shall now deal with a few of the more important symptoms and incidentally refer to the various types.

TYPES AND SYMPTOMS.

Raymond de Chalin, of Avignon, and Boccaccio, among others of that period, have left us a few remarks on the symptoms presented by the disease as they saw it.

John of Burgoyne was perhaps the first Englishman to make notes, though they are very meagre and of no value. The Bishop of Aarhus, in Denmark, on the other hand, compiled a valuable treatise on Plague in the fourteenth century.

Dr. Gilbert Skene of Edinburgh was really the first to leave any records showing sound thought and reflection.

Le Baker, who lived earlier than Skene, describes the outbreak in Oxford. He draws particular attention to the hardness and dryness of the swellings when opened. (As it was the custom to excise most of the buboes at once, in those days, these two characteristics were to be expected.) Sir Andrew Faulkner, Surgeon-in-Charge at Valetta, in 1813 writes in the Edinburgh Medical Journal of that date an extremely interesting treatise, in which he calls attention to the eccentric nature of the disease, its varied symptoms, its method of spreading.

Among the symptoms Faulkner notes nausea, vertigo, prostration, rapid pulse and injected eyes; and he says that thirst was not a prominent feature even with high fever.

Mason Good in 1836 said that plague and yaws were in some respects related to one another; he called them "Carbuncular Exanthems." In this he was wrong.

Bitter in 1896 said that there was a similarity between Plague and Anthrax; but it is difficult to trace any relationship between these two diseases.

The incubation period of plague is said to be anything between a few hours and fifteen or twenty days. Much of the discrepancy connected with these statements is due to the unreliability of native observers. Those who have worked in India well know that patients and their friends, as well as many hospital assistants, will answer a question according as they imagine they are going to please the questioner.

In my own experience I have never known the incubation period to be less than two days or longer than twelve. The average is from four to five days.

In the first years of the present outbreak in India and in China several symptoms were described as being peculiar to and characteristic of plague. And in some of the recent text-books the mistake is still continued. We read of "the characteristic plague tongue", with its white

furred surface,its red sides and tip. That this appearance is often to be seen there can be no doubt,but that it is characteristic of plague I should be sorry to admit.

Of much greater importance than any of these so-called landmarks is the absence of any definite symptoms in a great many cases.

It must never be forgotten that a plague-infected person may show the most transient and apparently trivial signs of what in a few hours is going to end his life. In an ~~ed~~ endemic area,or during an epidemic,no symptom however mild and momentary should be overlooked.

Among my notes I have records of several cases,from which I take two to emphasize this point.

Case 1. Sepoy,aet.about 30. Nasik,1901. History. Went on duty at 7.0 A.M.,feeling perfectly well. About 10.0 A.M.,he said he suddenly felt giddy and almost fell down. Reported, and was sent to Civil Hospital. Seen by Chief Hoppital Assistant,who found nothing warranting his detention.

Sepoy went home and kept quiet. I saw him at 9.0 A.M. next morning on my rounds. He was huddled up on charpoy. Eyes watery and slightly injected. Pulse weak,fluttering and very

compressible. Temperature 102.5. Right groin painful,hot,but could not feel any glands swollen. Was given hypo.of Strych. and removed to plague hospital.

11.0 A.M. ~~same~~ same day: dazed,but if spoken to sharply,replied. Pulse steadier,respirations increased. Tongue flabby and furred.

Died at 6.0 P.M.

Case 2. April 27th.,1906. Mysore.

Sepoy,Punjabi,aet.about 25.

History. Was seen by me on parade at 8.0 A.M.; had no complaint to make.

10.0 A.M.,on returning to hospital,I found the man coming away. The H.S.had seen him and reported nothing wrong.

On being questioned,he said that half-an-hour after inspection he felt giddy: he was sent to hospital. Examined by me,he seemed all right; but was taken in for observation,much to his annoyance.

1.0 P.M. Temperature 103;pulse 140,soft,very compressible; tongue clean; no signs of buboes or tenderness; mind perfectly clear.

Died at 3.30 P.M. same day.

Autopsy: highly injected intestines,mesenteric glands swollen,spleen liver and lungs congested. Smears from all organs and heart's blood showed bi-polar staining bacilli.

Here then are two cases,in totally different parts of India,and with an interval of five years between them. The first case was distinctly bubonic in type. An autopsy on this case was refused,but the man had a very apparent bubo. He had been away on leave for ten days,and two days after his return he was taken ill. Had he not been a sepoy,he would probably not have come under notice so early,if at all. On inquiry,it appears that he was staying with a brother in his village,who had returned from Bombay about a week previously; and this brother died the day after the sepoy resumed duty at Nasik.

In the second case,rats had died in the police lines about ten days before,and there had been cases of plague in some huts about half a mile away.

I have notes of many other cases which at first only showed perhaps slight headache,nausea,giddiness,or a sudden looseness of the bowels; but within twenty-four hours the patients were either dead or beyond recovery.

This shows,therefore,that nothing is trivial during a plague epidemic.

My experience teaches me when dealing with plague to distrust ordinary rules which might guide one to a diagnosis. Certain prevalent symptoms,however,must be mentioned.

Hiccough. This is a most distressing symptoms,and in some epidemics it was much more prevalent than in others. I never saw it in a pneumonic plague case,but always in severe bubonic cases,and never before the third day. In the Karachi epidemic of 1900 over fifty per cent of the cases suffered from hiccough.

Cardiac Failure. In plague,no matter the type,we are always confronted with this most serious symptom,to which may be attributed the very high death-rate in this disease. I am fully convinced,that if it were possible to impress the native patients and their friends with the always-present danger of heart failure,and so gain their co-operation in the treatment of their cases,the death-rate in India would be materially reduced. During the Karachi epidemic I was given more or less a free hand. My hospital staff consisted of eight highly-trained English nurses, four well-trained native hospital assistants,and as many ward boys as I asked for. Day and night there were always three nurses and two assistants on duty. In previous as well as in subsequent epidemics the death-rate varied between 70 and 80 per cent among hospital-treated cases. During the one in 1900 the death-rate was 47 per cent. This reduction was entirely due to the constant attention given to each case by the staff. In other epidemics in which I carried out almost the same medicinal treatment,

the death-rate ranged between 60 and 70 per cent. And the increase was due to its being impossible to give the same supervision to the cases. Time and again patients and their friends were warned of the certain danger of heart failure, and were told that to sit up or get out of bed would end fatally. They all promised to carry out the instructions. But on returning in a few minutes, you would find the patient being held over the pan on the floor. On more than one occasion this has led to the patient dying in his friend's arms. Among Europeans and Eurasians the death-rate is much lower; but with them one has co-operation, and so long as the patient is sensible he will lie perfectly quiet, thus giving his heart every chance. Other symptoms, such as sleeplessness, will be considered under the head of treatment.

Some writers in the beginning of the nineteenth century recognised at least two forms of plague; one a fulminating, another a bubonic type. Their fulminating type must have been the septicaemic or pneumonic kind of today. And strictly speaking there are only these two kinds of plague — bubonic, which is by far the commonest type, and pneumonic. Some writers mention a third, septicaemic, and even a fourth, ambulatory. The list could be still further increased if we are to be guided by prominent symptoms. At one time, I was inclined to think there was an alimentary type, because in some epidemics one came across a sequence of cases in which no apparent

buboes could be found externally. The main features in these cases were acute vomiting, violent diarrhoea, and death within thirty hours. On making autopsies, I have always found great infection of the whole alimentary canal, and invariably an infection of all the lymphatic glands, those most affected being the mesenteric ones. Some observers called this "Choleraic Plague". There can be no doubt that these patients died from an acute intoxication, a septicaemia, but the ~~ate~~ autopsy always showed "internal buboes."

BACTERIOLOGY.

Yersin and Kitasata were the first to isolate the plague bacillus. This they did independently in 1894.

Man does not show the highest degree of susceptibility to this organism. Though classified among the septicaemic bacilli, it is not truly so for man. He occupies the middle position. A highly susceptible animal should show no local reaction after inoculation. Man does; but the interesting point is, that the ~~re~~ reaction is not at the seat of inoculation; it occurs in the nearest group of lymphatic glands.

In the earliest stages of a bubo the plague bacillus can be obtained in pure culture by puncturing with a hypodermic needle. As the disease advances

the bacillus becomes altered in shape and loses its virulence. When suppuration has been fully established, you will not find the plague bacillus in the bubo, or, if so, in such altered forms as to be unrecognisable. This is of importance and will be referred to later on.

In pneumonic cases the sputum is full of plague bacilli, and four times out of five a pure culture may be obtained from it.

I have never been able to isolate plague bacilli from the faeces, and I question very much if faeces from a plague case are infectious. All experiments which I made with the view of infecting guinea pigs and rats by rubbing in faeces into the abdominal walls proved in my hands negative. Also by feeding rats on portions of meat rubbed up with faeces I failed to get any results.

I know that there are some who say that faeces and urine carry the plague bacilli, and are therefore sources of infection. I have repeatedly examined both faeces and urine for plague bacilli, but have never found them. In rats, dead from plague and with fully distended bladders, I have made cultures and slides from the urine, but always with negative results.

Blood. Gordon says that he has been able to show a terminal flagellum in many of the bacilli. Here too I have failed to find anything resembling a flagellum. He also says that it is encapsulated, especially in specimens from blood. Again

I am unable to agree with this. Slides showing bi-polar stained bacilli surrounded by a capsule have been sent to the laboratory for confirmation. These have turned out to be ordinary pneumococci. At times, and in cover-glass preparations made from gelatine growths, one sees a kind of halo round the bacilli, which might in a hurried examination be passed as a capsulated plague bacillus; but if the slide is washed, after fixing, in dilute acid solution, the halo will not be found.

The growth of plague bacilli on glycerine agar shows a constant character which so far I have not seen mentioned. If the surface of the growth is lightly touched with the point of a platinum needle, and then very gently raised, the growth may be drawn out into sticky-looking, fine, delicate threads. This is invariable, and more marked in young growths. The pneumococcus and the staphylococci do not show this stickiness. Yersin, quoted by Manson, says that "if grown on gelatine peptone, some parts of the colony grow more rapidly than others; and that if guinea pigs are inoculated from these more rapidly growing areas, it will be found that the virulence of the bacillus is diminished, and that on making sub-cultures it soon ceases to be fatal to guinea pigs." This of course is very interesting,

that in one and the same colony there should be be two rates of growth. The loss of virulence will take place in any growth if sufficient sub-cultures are made, but the virulence can be renewed by passing the bacillus through a series of rats or guinea pigs. "Frequent passage of an organism through animals may lead to a permanent or temporary increase in its virulence, or to a diminution of the same." (Sternberg) In so far as this latter fact may throw light on the natural decline of many epidemics it is worthy of note.

INTERACTION OF VARIOUS ORGANISMS.

In many infectious processes we may find two or more organisms in symbiosis. Experimentally it has been proved that a nonpathogenic organism may be rendered deadly if another equally non-pathogenic organism is introduced at the same time. Or again, the virulence of an organism may be "augmented or diminished by the concurrent inoculation of another one which itself may or may not be pathogenic." (Sternberg)
I have underlined the word diminished for a purpose.
I have stated above that in the earliest stages of a bubo you can get a pure culture of the plague bacillus. As the disease progresses, the glands soften and eventually break down. When first infected, the group of lymphatic glands are in a normal and healthy condition. The local as well as general action of the toxic material

produced by the plague bacilli gradually or rapidly lowers the vitality of the infected area, and this loss of vitality is favourable to the entrance of pyogenic organisms. The plague bacillus does not belong to the pyogenic group of bacteria. The presence of one of the pyogenic bacteria seems to be inimical to the life of the plague bacillus; and as the pyogenic organism increases in number with the formation of pus, so does the plague bacillus disappear. I have repeatedly been able to trace by hypodermic punctures the truth of the above statement. Starting with a pure culture, the bacillus has passed through the various forms of disintegration until the pyogenic bacteria alone held the field and a suppurating bubo free from all traces of plague bacilli took the place of the original condition. In the "Edinburgh Medical Journal" in 1806 there is a a very interesting note made by Baron Larrey, Chirurgien en Chef de l'Armée de l'Orient. He says:- "Before the army left Syria a great many of the soldiers were attacked by plague. It seldom attacked wounded men, and scarcely an instance occurred of its attacking a man while his wounds were in a suppurating condition, though many of them became infected as soon as their wounds were healed. "

Baron Larrey noted clinically a fact for which a hundred years later we are able to give a

scientific reason.

Round this many suggestions for treatment might be given, and under that heading I shall have to mention one method which was tried for a time.

There are many epizootic diseases whose bacilli may be easily mistaken for that of plague. The staining characteristics of plague bacilli are well known — very easily stained, decolourised by Gram's, and showing the bi-polar character. There is one other characteristic by which it can be distinguished from all other bacilli, namely, the stalactite growth in bouillon to which some oil or butter has been added.

COMMUNICABILITY TO ANIMALS.

In 1904 Simpson stated that fowls, ducks, pigeons, and other farm-yard feathered kind were susceptible to plague. He has been quoted by writers in many parts of the world on this point. In 1903 I had failed to convey plague to pigeons, and when I saw his statement in one of the Indian dailies, I decided to repeat my experiments. These I carried out through the latter months of 1904, and I repeated them all again in 1905, and some with pigeons in 1906. I worked entirely with fowls and pigeons which were purchased in the local bazaars or which I reared in my own compound. Ducks and turkeys I did not experiment on; so I am unable from personal evidence to make any statements. But when at the end of 1905 I was in Bombay, and in conversation with Colonel

Bannerman I mentioned the question,he told me that experiments carried out at Parel had proved negative,but that the matter would be gone into again. The following gives my own experience.

Pigeons. Six birds,male and female,were placed in a specially-made hutch. This was wired in, but the birds had ample room for flying about, and the floor was the natural earth. The birds were free from halteridium,as well as other infection. Each bird got an intraperitoneal injection of 1 cc. of a 24=hours' growth of plague bouillon. One rat and one guinea-pig were given 1/4 c.c. of the same bouillon. Both animals died within forty-eight hours. From them a pure culture of plague bacillus was obtained. At the end of a month the six pigeons were apparently all right. Cover-glass preparations and agar tubes from their blood showed nothing. They were then given 2 c.c. of a virulent culture. The controls died within 36 hours. At the end of three weeks the birds were still all right and had commenced to breed. For upwards of twelve months these birds were kept under observation,receiving from time to time intraperitoneal injections of plague bouillon. At the end of this time the six were killed and examined. I could not discover anything abnormal with any of the organs,and all tubes inoculated from them were sterile.

In another hutch six more pigeons had been placed at the same time as the above. These were fed on boiled gram soaked overnight in plague bouillon. At times the gram was rubbed up with portions of infected spleen. After twelve months these birds were killed and appeared normal.
In all my experiments on pigeons I never once saw one of the birds show any signs of illness.
Fowls. The same experiments were carried out with fowls,but only three were taken for each batch. The results were the same as for the pigeons. The Indian fowl will eat anything that comes in its way; so I had no difficulty in feeding those under experiment on quantities of plague-infected organs.
Similarity between B.Pestis and other organisms found in septicaemic diseases in animals.
The following are some of the chief septicaemic diseases in animals whose bacilli may be confused with that of plague.
A. Septicaemia Haemorrhagica. Similar to fowl cholera,rabbit septicaemia,and hog cholera.
This is a widely-distributed bacillus,found in putrifying infusions and impure river water,and has been isolated from the salivary secretions of man (Baumgarten).
Morphology. Short round ends,in pairs or chains, bi-polar staining decolourized by Gram's method; non-liquefying. Best temperature for growth between 35°and 37°C.

In gelatine,in two or three days small white, finely granular round colonies appear,irregular in outline,and yellowish by transmitted light. In agar,it is a streak,limited to line of inoculation,finely granular,semi-transparent,with irregular margins.

In peptone,it gives reaction for phenol and indol. Pathogenic to pigeons,chickens,mice,sparrows and rabbits. Rabbits are very susceptible,and die within 20 hours. The post-mortem appearances are similar to those seen in plague. In rabbits and fowls it may run a more or less protracted course,if inoculated with a small amount of an attenuated culture. (Sternberg) Smith and Moore state that an ~~attentu~~ attenuated variety of bacillus belonging to the group of swine-plague bacilli and not distinguishable from them inhabits the mouth and upper air passages of domesticated animals,dog,cat and cattle.(Smith)

B. Bacillus of cholera in ducks. Pathogenic for ducks,fowls and pigeons. (Cornil and Toupet,1888)

C. Hog cholera. Bacillus bi-polar,staining decolourized by Gram; motile; pathogenic for rabbits,guinea pigs,pigeons and mice; may occur as an acute and fatal septicaemia; post-mortem appearances similar to plague.(Sternberg)

Bacillus of Swine Plague (Marseilles). Rounded ends,bi-polar,staining decolpurized by Gram; motile; not liquifying; distinguished from hog

cholera by producing indol and phenol reaction in peptone. (Sternberg)

D. Bacillus Septicus Agrigenus. Nicolaier got it from soil which had been manured. It is like bacillus of fowl cholera and of rabbits, and is no doubt a variety of this, but longer. Shows at times bi-polar staining, non-motile. (Sternberg)

It will be seen from the above that some of these bacilli at any rate may be mistaken for that of bubonic plague. I do not contend that this has been done in China, but other experimenters in India have been unable to infect pigeons or fowls with bubonic plague. With several of the above bacilli common to fowls and pigeons, there may be the possibility that some of them have been confused with that of bubonic plague. If this is not the case, then it would appear that in China and other parts of the world fowls and pigeons are susceptible to plague, while Indian birds of the same family are immune; Or, that all the experiments carried out in India by independent observers were faulty.

The matter is one of no small importance; for, apart from the necessary loss to the owner if the birds are destroyed, there is of course the more serious question of infection to human beings.

PREDISPOSING CAUSES, AND SOURCES OF INFECTION.

Turning now to the causes of infection, one cannot but be struck, as also in considering other aspects of the subject, by the great variability shown throughout each and every epidemic. All statistics and theories built up on them may in the next ~~epidemic~~ one epidemic be overthrown, if not in the same district, then almost certainly in the adjoining one. It is this fact, this lack of methodical sequence, that raised so many obstacles in the earlier years of the pandemic.

There is in my opinion no one particular predisposing cause which may be pointed to as peculiar to plague. Insanitary conditions are the most important, in that they harbour vermin; but time and again I have seen the most insanitary portions of a town passed over, and those in which, if the rule had been constant, plague should not have appeared, badly infected.

When the disease first broke out in Bombay in 1896, it passed over the most insanitary districts, attacking those inhabitants who by caste laws were compelled to keep themselves cleanly. In Poona, during the 1900 epidemic, the same thing happened, as it was among the better class people that the disease played greatest havoc. But in the following outbreak in the same city it raged through all quarters.

In Sholapur it commenced in the poorer localities and spread to the better ones. But the most

striking example of epidemic after epidemic occurring in a large area which was under stringent sanitary laws is that of the Kolar Gold Fields, Mysore.

As I shall have to refer to this district on several occasions, I propose to give a somewhat detailed account of the mining camp, or rather series of camps, for there are seven or eight. The following description deals only with the native camps. The European quarters were quite separate. The camps commence on the north end of the "Fields" and stretch in a slightly curved line for over nine miles, ending in the south end. The average width is roughly half a mile. Between each camp, according to the various mines, there is about a quarter of a mile, but the mills and working sheds etc. run continuously from one end of the Field to the other. In some cases they come close up to some of the coolies' huts, but as a rule they stand well away from them. East of these sheds and running north and south is a wide road, and east again of this, but standing on much higher ground, are the European bungalows and the large Mining Hospital.

Each camp is now made up of separate huts arranged in rows with lanes about twenty feet wide running between each row. Cross lanes ten feet wide separate each hut. When I first took over charge of the laboratory and plague work, all the huts were built of ordinary tattie matting nailed to

upright posts. If plague broke out in one of these, the whole place was burned down. In time I had all the huts built on the following plan. Four corner poles twelve feet apart and twelve feet above the ground were fixed in position, to form a square, with intermediate poles between. Two sheets of corrugated iron were then nailed lengthwise on to a frame, and these frames were ~~are~~ screwed on to the lower part of the poles so that a foot of the iron was below the surface of the earth and about four feet above. The upper part of the hut was then closed in by tattie mats nailed on to frames, which in turn were screwed to the posts. The roof was made of corrugated iron meeting at a centre angle. The floor was native soil, into which had been mixed some sand which had come through the cyanide mills; to this was added some lime, and the whole was beaten down hard. In less than ten minutes a hut could be taken to pieces. Over each camp there was a head sanitary inspector, usually a discharged English soldier, or a Eurasian who had a sanitary certificate from Government. Under their control from four to six sub-inspectors looked after each camp, and gangs of sweepers.

During a plague epidemic each hut was visited daily by a sub-inspector. No rubbish was allowed to accumulate, and all refuse was burned in large incinerators. Numerous latrines were provided, and no nuisance was permitted under heavy fines.

Commencing at opposite ends of alternate rows of huts, the floors were dug up every two months all the year round. To do this it was necessary to remove the lower frames with the corrugated iron. Ample water was supplied by means of stand-pipes. All rats were trapped or poisoned; Liverpool rat virus was put down. There was a constant watch kept for them; they could not be exterminated from the numerous sheds, but they got little peace in the lines of huts. The average number of coolies in residence in this area was about 20,000. Another 15,000 lived in villages from one to five miles round the mines. These came in for work daily. All these villages were under the charge of two Mysore Government native doctors, and they had six sanitary overseers constantly inspecting. As soon as plague broke out, the staff was increased and extra help given by the mining doctors. Notwithstanding all these precautions, there has been a yearly outbreak of plague on the Kolar Gold Fields. Some years it was not quite so bad as others. But often, when there seemed no possible chance for its spreading, it would run through a camp with exceeding rapidity.

There could be no question of filth or insanitary conditions playing a part in these epidemics. If plague broke out in a camp, every hut in that camp was at once taken to pieces, the floor dug up, and the frames soaked in carbolic or other disinfectant. If the tatties were old, they were

burned. In the adjoining camps, the entire contents of each hut had to be put out in the sun for two hours every day until I gave orders to the contrary. How then did it spread? Let us consider different possible causes for this.

Effect of Climate on the disease.

It is generally accepted that a mean temperature above 85°F. or below 50°F. is inimical to plague. Simpson says that anything between 56° and 75°F. is favourable. Lane in "Modern Egypt", written in 1833, says: "El Khamaseen or hot southerly winds raising the thermometer to 95°F. are dreadfully oppressive even to the natives. When plague visits Egypt it is generally in the Spring, and the disease is generally most severe in the period of the Khamaseen. Flies are abundant in Spring, Summer and Autumn, and in the cooler weather fleas are extremely numerous. It seldom extends far above Cairo, and is most common in the marshy parts of the country near the Mediterranean S. Browning Smith, in the "Indian Medical Gazette", speaks of " a gradual beginning and a period of activity in the autumn, a lull or decrease in the winter, increased activity in the spring, a gradual fall to absolute freedom in the summer."
This relates to his experience in the Punjab.
In Bijapur, in 1902, there was a sharp outbreak during the months of March, April and May, the three hottest months in the year. With the advent of the monsoon it died out.

The present epidemic in Harbin and the sur - rounding country is proof that it can rage through the depths of a Siberian winter.

Human Influences.

Bitter,in the "Report of the Plague Commission",1896 - 7 -8,says: "In all simple cases of bubonic plague the bacillus must have gained admission through some minute lesion in the skin at one or other extremity."

This is an accepted fact,and purely bubonic cases without supervening septicaemia,or running a short and fatal course,are not a source of infection.

I have made two exceptions,for it seems to me that if we are to accept the flea theory,these two exceptions must be taken into account. This point will be further discussed under that heading. In all my hospital records I cannot find one in which an attendant was infected directly from a simple uncomplicated case of bubonic plague. In such cases it is very difficult to find the bacillus in the peripheral circulation. I have repeatedly examined the blood of hospital patients, both microscopically and by taking 1 c.c. from some vein and inoculating agar tubes. In what are spoken of as septicaemic cases I never failed to find bacilli,but in simple bubonic cases I rarely got any positive results,unless the patient was dying,but even then the number was not so great. With the pneumonic form of plague we are dealing

with a highly infectious and contagious disease. The patient's sputum teems with plague bacilli. In a hospital, where precautions are taken, the disease does not get the same chance of spreading, but in towns and villages one such case will be sufficient to create a serious epidemic. The blood in these cases is full of bacilli, and here again vermin must play an important part. And it must never be forgotten that one pneumonic case is much more serious than a dozen bubonic ones. In Karachi, Poona, Nasik, and Mysore I lost one, two, or more of my ward boys, and there can be no doubt but that they became infected by being in close attendance on some friend who was suffering from pneumonic plague.

Pneumonic plague sputum is highly infectious. I have on more than one occasion rubbed sputum from one of my cases into the shaved and punctured abdominal wall of a rat or guinea pig, with the invariable result of death within three or four days from plague. Again, I have rubbed boiled gram up with plague sputum, and fed rats and guinea-pigs on it, with the same fatal results.

If it is thus so infectious to rats and guinea-pigs under these artificial conditions, what is to prevent its infecting grain and other food-stuff in the rooms where the patient is confined? And if a rat eats some of this, it will in turn become infected and so start an epizootic.

It is in this way that man becomes an important factor in the spread of plague. But there are other agencies which are of far greater importance and it is chiefly to them that one must give the greatest attention.

The way in which plague will spread through a town or village, or from one ~~twon~~ town to another, is at times most puzzling. A walk through a plague-infected district and a note made of the marks on the wall outside the doors of houses in which plague has occurred will be sure to make the observer wonder if there can be such a thing as "place infection." In the Karachi epidemic of 1900 the disease broke out in the same chawl and on the same floor of the chawl as in the previous year. It then spread through that district for over ten days, following almost the line of progress of previous years. So marked was this sequence of events, that Colonel McCloghry, I.M.S., said to me: "Within two days or so we should hear of cases in the Sudur Bazaar." Next day a case was reported, and so it went on from quarter to quarter, until it died out in the same locality as in the previous epidemics. Why is this? Do rats follow some natural law of which we know nothing? Compare the plague marks on houses. You will notice some walls with as many as ten or a dozen circles and dates, and you will further be struck by the fact that these follow upon each other at almost equidistant periods of time. Is this

interesting coincidence a mere matter of chance? Does chance play so important a part in a dozen different places in India? These are questions which may be answered in some slight degree when we come to consider the part played by rats and fleas in the transmission of plague.

Rats and Fleas.

In ancient times the inhabitants of plague-stricken districts learned to connect a high mortality among rats with plague. Strabo quotes Posidonius as ~~at~~ stating that the Iberians used to move their camps or villages when they noticed that rats were dying in any appreciable numbers. In some of the old purans in Indian literature, one reads of warnings against rat mortality. The honour of first formulating the Flea Theory is given to Dr.Simond.(Browning Smith,in the "Indian Medical Gazette".) Like every new theory,it received by scant notice at first,except from one or two.

In 1903 I made a series of experiments to prove the connection between rats and fleas in the transmission of plague.

Several rats were caught in my own compound,and in neighbouring ones. There was no plague in Mysore at the time,and there had never been any in the district from which these rats were taken. Over a dozen rats were caught and carefully examined for any signs of plague or any glandular disease. The results were all negative; so it was assumed the others were also free from infection .

The rats were placed in specially-made boxes and fed on boiled gram. After a month some of them had littered, and so increased the stock. At the end of six weeks plague broke out in a village about four miles distant. Two rats were placed in a spe separate box and kept for ten days; then one was removed and placed by itself. It was fed on small portions of human spleen taken from a man who had died the same morning in the plague hospital.

July 26th., 1903. Glycerine agar tubes were inoculated, also oiled bouillon. Cover-glass smears from spleen shewed bi-polar stained bacilli.

July 28th. Rat huddled up in corner of box. Died during the night.

July 29th. Post mortem. The muscles did not show serious infiltration — one can always find this infiltration if subcutaneous inoculation has been carried out. The whole intestinal tract was greatly congested, the serous covering of abdominal organs seemed moist and glistening, there was a half-inch intussusception in the small intestine. Bladder full of clear pale urine.

Liver, spleen, lungs congested, heart dilated and full of blood.

Cover-glass smears from all organs showed bi-polar bacilli.

Cultures were made from heart's blood, and spleen. The growths on all media were identical, that is, of human as well as rat's organs.

The control rat did not die.

A third rat fed on portions of the dead one died in forty-eight hours.

A fourth rat inoculated with culture from human spleen died in three days.

A fifth rat inoculated with culture from first rat died in thirty-six hours.

It was thus evident that rats could become infected if they ate portions of human or animal organs from plague cases.

Early in September a rat was found dying in a native workshop on the north end of the Fields. This workshop was several hundred yards from the coolie lines, and at that time there was no plague in that camp. The previous year there had been an outbreak in the neighbourhood, but all the huts had been pulled down and the camp moved to fresh ground half a mile away. No one lived in the work-shop. The dying rat was placed in a tin box and sent up to the laboratory. On examining it I found that it was swarming with fleas, P.Cheopis. Through a small hole in the lid of the box 1 in 50 carbolic solution was poured in. After two or three minutes the box was well shaken, and I then picked out twelve fleas. The rat was drowned. The fleas were pressed lightly between blotting paper and then put in a Petrie dish. In a few minutes they began to show signs of life; so I picked out six and placed them in a cage covered with fine gauze. In this cage I had previously put a rat from my old stock.

Of the remaining three fleas, two were placed in a test tube with a glycerine agar slope, the third was crushed between fine forceps and a platinum needle, and stroke inoculations were made on glycerine agar. After thirty hours the inoculated agar showed a slightly mixed growth, but it was not difficult to isolate plague colonies from a plate culture. The tube with the two live fleas never showed any signs of growth. From the isolated colonies agar tubes were inoculated and a pure culture of plague bacilli obtained. Rats and guinea-pigs were inoculated from these, and they all died from plague after a few days. The rat in the gauze-covered cage was found dead on the sixth day. Post-mortem examination showed that it had died from plague, and there were five small petechiae on the inner surface of the abdominal skin, near the region of the thorax. The axillary and inguinal glands were congested, and from them as well as from the heart's blood I was able to get a growth of plague bacilli.

Similar experiments were repeated again and again during the next three years, and always with the same results, this establishing the connection between rats and fleas.

In 1905 I carried out the following experiment. A rat was placed under a bell-jar, and a

piece of blotting paper saturated with chloroform was then pushed in. All the vermin on the rat fell off. The rate was then inoculated with five minims of a plague bouillon,and was placed in a large wooden box half filled with earth. In this box there were ten other rats,which had lived there for over six months. In a little over forty-eight hours the rat was found dead on the top of the earth. P.Cheopis were found on it. Post mortem showed the site of inoculation.
Within three days four more rats died; then a couple of days passed and three others died. P.Cheopis were found on all of them,and small petechiae along the inner surface of the thoracic abdominal skin. A week passed,and as there were no signs of any more dying,traps were set and the three remaining ones caught. The box was then well sprayed with carbolic and the soil was turned over. The rats were put back,and six weeks later were still alive. Five fresh rats taken from the old stock had fine wire wound round their fore paws and were then let loose in the box. After the space of three weeks,one was found dead. It had died from plague. A week passed and two more died. Then one of the original rats was found dead. All had died from plague. Plague-infected fleas were found on all of them,and also on those which had not died. One of these was killed,but I could not find any petechiae or any signs of infection. At the end of six months,

it was evident that they had been breeding,as young rats were seen on the surface,but there were no more deaths from plague in that box.

It has been conclusively proved that a rat infected with plague,but free from vermin,will not infect other rats living in the same cage if they are likewise free from vermin. Therefore there can be no doubt that the main link between rat and rat ~~id~~ is the flea. What then is the link between rat and man? Is it the flea alone, or are there other insects? And is it only the rat flea,or are there other fleas concerned? This brings us to the question of other animals. As previously stated,I do not consider that pigeons and fowls are susceptible to plague; so that they need not be taken into account. I had to examine many fowls in India,which had been sent to the laboratory for "cause of death",in which I could not find any bacilli in the blood or elsewhere; but recently I had the opportunity of inspecting some fowls in the "Welcome" laboratory in Khartoum under the directorship of Dr.Andrew Balfour,and I feel confident that the Indian fowls must have died from a spirochaetosis - a subject which Balfour has worked out during the last three years.

Cats. Of other domestic animals which call for special notice the cat stands out prominently . Buchanan and others strongly advise that cats should be kept as a means of keeping down rats,

in other words,as a prophylactic measure. There can be no doubt that anything likely to diminish the number of rats in a house or village must be taken into serious consideration. But is it quite certain that cats do not themselves contract plague,if not in a virulent form,perhaps in a mild and chronic one,but still one which may be a source of danger to the occupiers of the same house? Cats swarm with fleas at certain periods of the year. Will the rat-flea live on cats? May the cat not be the means of conveying the infected rat-flea to man? All these are questions which must be looked into. We know that the plague bacillus is highly infective to rats,that it fulfils all the requirements of the septicaemic bacilli with them. May it not occupy the other end of the scale with cats? There is no fatal septicaemia,no high mortality to attract attention,but there may be a mild local infection,and if the dose is large enough,death will follow. I have examined several cats from plague-infected villages in India. It had been noticed that they were apparently out of sorts,and that they lay about the compound for some days. On three or four occasions I found marked enlargement of the cervical glands,and a caseous condition in many of them. I was told by the natives that one or two cats had died during previous epidemics,but not many.

Two cats were obtained from one of the villages and the following experiments carried out.

They were both fed on portions of plague-infected rats recently dead from plague. For two days they seemed all right. On the third morning both were found lying on their sides, and crying out from time to time as if in pain. Towards mid-day they began to have loose motions, and lay with their legs drawn up. They ate a little food, and lapped milk.

Fourth day. Huddled up, quiet, no motions, breathing rapid, did not take any notice when spoken to.

One died during the night, and the other soon after 10.0 A.M.

Post mortem. No infiltration of tissues, no fluid in peritoneal cavity. Small intestine slightly congested; liver highly so, and covered with yellowish sago-like spots; spleen same as liver; lungs very much congested, especially the left in one of the cats. There was a quantity of sanguineous fluid in the pleural cavities; heart full of dark blood. All the mesenteric glands were congested, and those at the root of the neck were enlarged and congested.

From the lungs and heart's blood I got a pure culture of plague bacilli, which killed rats and guinea-pigs in two days.

It is true that I had given the cats highly infected organs to eat, and had fed them on such for over two days, but there was no doubt but that both animals died from plague. And, if a cat, in

the ordinary course of events, were to kill a rat within a few hours of its death from plague, might not the cat itself become more or less infected and therefore a likely source of danger? I do not desire in any way to minimise the value of cats as rat-controllers, but there is the question in my mind whether cats may not be a means of carrying on the epizootic. This of course would not account for the continuance in every place, as in some districts cats are not kept; and it has been put forth as an argument in their favour, that in those localities in which cats are common household pets plague does not exist, or at least not in epidemic form.

Snakes.

The only other animal which I shall mention is the snake. I do so because I have during the last few days received a cutting from a London Daily, in which Dr. Sambon advocates the keeping of these animals. I kept three cobras in 1905, and tried to inoculate them with plague. In this I was unsuccessful. The question of harmless snakes being kept as rat-catchers is one which probably will not appeal favourably to the public mind.

PROPHYLAXIS.

Every effort should be made in the direction of prevention, but that we are very far from successful the history of the past decade tells us only too clearly.

The difficulties of carrying out any scheme on a large, or for that matter on a medium scale in India can only be fully appreciated by those who know the country and the antagonism shown to all innovation by the vast majority of the inhabitants. The question may be discussed under two heads: First, what are the steps one should take to prevent plague from entering an uninfected country, or an uninfected city in an already infected country? Second, how should one prevent its spread through a town which has become infected?

Under the first head, the strictest quarantine rules will have to be enforced: there should not be any half measures. Every avenue of ingress must be watched. All ships arriving from infected countries should be specially dealt with, and anything likely to harbour rats or fleas must be suitably disinfected. There should be no night unloading, in case rats should escape to the shore, and all hawsers and chains must have guards placed on them, so as to prevent rats from crawling along them. The destination of all persons on board should be noted, and if thought necessary, their clothing, goods, etc., should be subjected to some vermin-killing process.

I think that it will be generally admitted that in the great majority of first infections it is man who is the carrier. He arrives from a plague-stricken place, and may have about him or in his

belongings plague-infected fleas. These fleas may infect a second or a third person,but the disease is not likely to spread unless rats become infected. Then,after an apparent lull,fresh cases break out and the town or village is faced with a severe epidemic.

That this,however,is the one and only way in which plague is spread,I am not inclined to admit,although no doubt the main channels of spread are rats and fleas. The rise and fall of the disease follows so markedly with the appearance and disappearance of fleas,that one is forced into accepting these insects as the chief source of infection both to man and rats. Nevertheless there are many cities in India in which rats and fleas abound,in which the habits of the natives are far from sanitary,in which the houses and streets are so clustered together that sunlight rarely finds its way in,and yet plague passes them over. I have in my mind the city of ~~Mdr~~ Madras,in some of whose quarters you will find ideal spots for the spread of plague. The outlying districts of Velore are badly infected with plague. There are three main lines of trains into Madras. Railway inspection is carried out,and has been for years; but anyone who imagines that natives from infected areas only travel by the railways is living in dreamland. Time and again plague cases have been found in Madras,but the disease did not spread. It is not for lack of filth,of rats,of fleas. Why

then?

In Sholapur,a city of over a hundred thousand native inhabitants,we find epidemics occurring in alternate years. There was a severe epidemic in 1900 in Sholapur,the first cases being traced to Poona; then after a few weeks the disease spread like wildfire,dying out by the end of May. In 1901,when I was Civil Surgeon there,I was on the look out for plague. March came and went,but the disease showed no signs of starting; but in April we had five cases in one week,scattered over the town,and all imported from Poona. By the end of the month we had three more imported cases,and eleven indigenous ones. No dead rats were found, and there was no epidemic; but it could not have been that all the rats had been killed off,for we trapped hundreds of them. The following year there was a sharp epidemic,to be followed by only a few cases in the next year. Therefore in an uninfected country or town particular attention must be paid to new arrivals from infected areas.

In a city which is already infected inoculation with Haffkine's serum should be made compulsory. Correctly speaking,this is not a serum,but a vaccine,and there is not the least doubt that it is protective. I have inoculated over 20,000 natives with it,and am fully convinced of its efficacy; but it is only protective for about six months,and must be renewed then. It is not only

protective,for should the person become infected with plague before the period of immunity is established,it seems to curtail the disease as well as minimize the symptoms. I have records of several cases which developed plague some two or three days after inoculation with Haffkine's serum; and as the interval between the date of inoculation and the appearance of the disease increased, so did the severity diminish. It is also well known that a pregnant woman who develops plague invariably aborts,the child if near full time being dead or dying soon after,and the mother seldom recovering. I have records of nine cases of plague in pregnant women who had been inoculated but who developed plague within a week: all aborted,but only two died,and both these cases showed buboes within forty-eight hours of inoculation.

Some contend that pneumonic cases are not infectious,but with this I am unable to agree; for in my opinion they are highly infectious.

In the "British Medical Journal",August,1910, Col.Buchanan,under the heading of "Cats as Plague Preventers",says:" If plague pneumonia is common, and if pneumonic cases are directly infective,we should expect to find epidemics among the attendants in a plague camp." And again,"A plague camp is one of the safest places during an epidemic."

Fortunately plague pneumonia is not common, but it is common enough to have necessitated its being placed in a type by itself. It by no means comes anywhere near the bubonic type in prevalence. "If it is infective, we should expect to find epidemics among the attendants." Now in my experience, which covers a great many hundreds of cases, I have several records of attendants having contracted plague in hospital, and in every case they got it through being in special attendance on pneumonic cases of plague. This occurred in Karachi, in Poona, in Satara, and I have seen cases in the plague hospital in Mysore.

One might argue that because hospital attendants do not get infected by ordinary pneumonia, it is not infective; but though we do not have special wards for ordinary pneumonic cases, I do not think that anyone will deny their infectivity.

Moreover, in my opinion, it is not a sound argument to say that because a plague hospital is the safest place during an epidemic, therefore pneumonic cases are not infective. In a plague hospital every precaution is naturally taken to prevent infection; the ~~walls~~ wards or cubicles are washed ~~are washed~~ down daily with antiseptics, all sputum is collected in proper receptacles, and should any fall on the floor or walls, it is at once removed and the ~~spot~~ well washed. The disease gets small chance of spreading. The picture in a native hut or house is very different. The patient expecto-

rates in all directions; his friends will wipe the tenacious sputum from his lips with their fingers,and then clean them on their garments, although the sputum is practically a pure culture of plague bacilli. Is not this a source of infection? No disinfectant is used. Moreover, when a patient is admitted into a hospital,the clothes is which he is brought are taken and placed in a tub of some disinfectant,he himself is wrapped up in clean sheets and is probably washed all over. Any fleas which may have been on him are drowned in the clothes. The chances of infection are reduced to practically nil. Is it fair to compare a plague hospital with the overcrowded quarters of a town or village; or apathetic,ignorant people with attendants who have been trained and are under a superior and highly-trained official?

In a district where plague has once been epidemic,I would insist on all cases of pneumonia being treated as pneumonic plague until the contrary is proved. On the Kolar Gold Fields,Mysore, the chief source of spread is man. The coolies know that if plague is found in their huts,all the inmates or suspects are removed to the detention camp for a week. They will therefore when they find a case try to hide it,and will go and sleep in some friend's hut,perhaps in another camp. We thus get scattered cases; but owing to the system of dismantling the huts,etc.,rats do not get any chance of breeding,and in fact few are caught in

the camps. Coolies from infected huts no doubt carry plague-infected fleas on their persons; and this would explain why in the face of every possible sanitary precaution we continued to get scattered cases of plague on the Kolar Gold Fields.

Occupation.

It has been stated by more than one observer that certain trades seem to predispose workers in them to plague-infection. It has been noted that grain-dealers show a larger percentage of cases than other people. This of course is accounted for by the fact that rats are more numerous in localities in which grain is stored. At the other end of the scale, workers in oil are supposed to be more or less protected. Faulkner, who was resident in Malta in 1813, states that those who worked in oil-shops were immune to the disease. He therefore strongly advised people to wear a kind of oiled silk as a protective measure. Recently, Purdy advocates inunctions of petroleum as a prophylactic measure. Fleas no doubt will not attack people whose bodies and clothes are always redolent of strong-smelling oil, and this has perhaps been noted by observers working in the recent outbreak round Harbin, for they have issued instructions to the staff to smear themselves with petroleum or to wear specially prepared under-garments of oilsilk material.

Treatment.

The treatment of Plague is one of symptoms. Cardiac stimulants must be given early, for the chief danger is heart failure. Strychnine holds the first place, but ether and brandy must be given as well. In 1904 I commenced to use Adrenalin: the effect is not prolonged, but in combination with strychnine it is most valuable and will tide a failing heart over a serious crisis.

Of other symptoms which call for special attention, hiccough is one of the most distressing. In some cases it is impossible to find a remedy. Small blisters placed at various spots over the epigastric region and frequently repeated are more efficacious than large ones. In combination with the blisters, I used drop doses of tincture of iodine in a wine-glass of water, given every fifteen minutes, or in severe cases every five. I seldom found these two remedies fail. Keeping the tongue fully extended for half a minute, and repeating this for five or six minutes, would in some cases be sufficient to stop the hiccough.

Buboes should never be opened in the early stages; the idea that removal of the inguinal glands does away with the source of supply is fallacious.

For delirium there is nothing better than hyoscine, should this fail, washing out the rectum with copious hot saline enemata will often produce a few hours' quiet sleep.

For the general treatment of plague there is no specific. In 1900 I tried injections of polyvalent anti-streptococcus serum. Out fe of fourteen cases of bubonic plague so treated there were nine recoveries,but as the cases were of a mild type,the probabilities are that they would have recovered in any case.

There is a drug,however,which I think is worthy of attention,namely,Collargol,an allotropic form of silver,recommended very highly in the"Medical Annual" for 1906,for cases of pneumonia. The following year I used it in some severe cases of plague in Bangalore,it being administered intravenously in a 1 per cent solution. The reduction of temperature and disappearance of delirium was so marked that it was determined to try it extensively; but I left India a few days after suggesting this treatment,and no further work was done by the native doctors in charge of the plague hospital. I am strongly of opinion,however,from the seven or eight cases in which it was used,that it is a drug which should be further experimented with.

Before leaving the matter of treatment, one other means should be mentioned of alleviating some of the symptoms,if not of curing the disease, which so far as I know has not yet been tried —I mean lumbar puncture. We know the great benefit of lumbar puncture in cases of cerebro-spinal meningitis,or of coma from Bright's disease,or

of high temperature and delirium accompanying some of the infectious fevers. Why, then, should it not give similar results in the delirium of plague? We remove a highly infected fluid, we relieve cerebral pressure, and we have at the same time a channel for the injection of any sera or drugs which we may desire to try. The danger of accident is nil, if the operator will remember not to remove too large a quantity of cerebro-spinal fluid at one time. From ten to fifteen cubic centimetres is the most that should be removed at one sitting, anything above this being apt to be followed with unpleasant results.

As all the foregoing remarks are based entirely on my own personal experience and experiments, there is little need for a general summary of my opinion. One point, however, calls for some attention. Are we not apt, when looking for the means by which plague is spread from rat to rat, from man to man, to say that the flea theory is the missing link, and that that being found and proved correct leaves us little more to do? That the flea is perhaps the chief source of spread and infection of plague, may no doubt be true; but that there are not some other agencies at work which have so far eluded our search, I am far from believing. There is still much to be found out as to the method of spread, the manner in which it is carried over the apparently quiescent periods; a means of

protection which will confer a longer period of immunity, and, lastly, some form of treatment beyond the rule-of-thumb cardiac stimulant.

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